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PROFESSOR RONALD ROSS, C.B., F.R.S., *President*, in the Chair.

THE DISTRIBUTION AND PREVALENCE OF YELLOW FEVER IN WEST AFRICA.

BY

SIR RUBERT BOYCE, F.R.S.

INTRODUCTION.

This paper is the result of investigations made during June, July and August, 1910, at the request of the Colonial Office. For the purposes of this investigation, I visited Southern Nigeria, the Gold Coast, Sierra Leone, and the Sierra Leone Protectorate.

I have had the advantage of considerable experience in yellow fever, having been present as a medical volunteer in New Orleans in 1905 during a very severe epidemic. Later in the year 1905, I visited British Honduras and the adjacent Central American Republics, at the request of the Colonial Office, and reported my investigations upon yellow fever in that part of the world.

In 1909, I proceeded to the West Indies for similar purposes at the request of the Colonial Office, and studied the yellow fever situation in Barbados, Grenada, St. Lucia, St. Vincent, Trinidad and British Guiana in South America. The present enquiry, therefore, forms the fourth

upon which I have been engaged. In many respects it has proved the most interesting and the most full of unexpected results, as I will now endeavour to show.

I.—THE STEGOMYIA IN WEST AFRICA.

The first question I put to myself was: What was the prevalence of the *Stegomyia calopus* in West Africa? Not merely whether it had been recorded, but in what proportion it existed. I soon satisfied myself that it was by far the most common mosquito of a very large number of towns. I have made careful house-to-house examinations and have recorded in very many instances the actual percentage.

In several towns the stegomyia was present in every yard, not only in one vessel, but often in every vessel which contained water. As has been noted before by others, as well as by myself, it was often present to the exclusion of other species of mosquito. There can be no question that it is the house or domestic mosquito, not only of the coast towns of West Africa, but also in every probability of a large proportion of the towns in the interior. BOUFFARD has drawn attention to its wide distribution in the Upper Senegal and Niger Territory; I have found it in Kenema and Coomassie; and OTTO draws attention to its distribution in Togoland. This mosquito is, therefore, one of the very commonest in West Africa, and when present is found in vast numbers. The following is a list of the places where it has been reported, and, in many instances, where returns of the prevalence have been made:—

Bathurst	Cape Coast	Coomassie	San
Freetown	Axim	Kulikoro	Dakar
Conakry	Elmina	Timbucto	St. Louis
Bo	Salt Pond	Ada	Gorée
Kenema	Quittah	Kita	Lagos
Secondee	Tarquah	Bamako	Calabar
Accra	Obouassi	Sissako	etc.

The condition of life and the water supply on the coast are all in favour of the great multiplication of *Stegomyia calopus*.

It is not a swamp, gutter or puddle breeder (as a rule), it is essentially the mosquito which breeds in *all receptacles* which happen to contain clean water, in no matter what quantity large or small,

especially small. Hence it comes to be the mosquito of all discarded tins, bottles, etc., etc. Hence it is most abundant where there are traders and is least abundant in the interior where there is less trade. The dirtier the town, the more receptacles, and the more stegomyia. It is not dependent upon the rains like the anophelines. It may be in immense numbers in the driest season because of the fact that it is found in artificial water containers as distinguished from natural puddles and other water collections. The rains, however, influence it, for more odd tins and bottles become filled with rain water. Increased trade and commercial activity provides an increase of odd water containers of all kinds, and, therefore, an increase of the stegomyia, and therefore a greater liability to yellow fever. As in every other part of the tropical world, with the advent of the white man, yellow fever appears in its severer forms. Drainage and building operations bring down malaria, but unless a controlled water supply is introduced, yellow fever makes its appearance in the non-immunes or increases as towns increase in size.

II.—HISTORY OF YELLOW FEVER IN AFRICA.

Having proved that the carrier of yellow fever exists in great abundance in West Africa, it now remains to discover whether yellow fever is known in Africa. I will be able to prove to you that yellow fever has existed for 100 years in West Africa, that is from the 18th century to the present day.

The history of yellow fever in Africa is that of the history of the disease in Central and South America and the West Indies. It has gone on *pari passu* with the development of western commerce.

The following is a list of the years in which it has been recorded up to date:—

YEARS IN WHICH YELLOW FEVER APPEARED IN SPORADIC OR EPIDEMIC FORM IN WEST AFRICA.

1807	1822	1837	1852	1867	—	1897
1808	1823	1838	—	1868	1883	1898
1809	1824	1839	—	—	1884	1899
—	1825	1840	—	—	—	1900
—	1826	1841	—	—	—	1901

YEARS IN WHICH YELLOW FEVER APPEARED IN SPORADIC OR EPIDEMIC FORM IN WEST AFRICA—continued:—

1812	1827	1842	—	1872	—	1902
—	1828	—	1858	1873	—	1903
1814	1829	1844	1859	—	—	1904
—	1830	1845	—	—	1890	1905
1816	—	1846	—	—	1891	1906
1817	—	1847	1862	—	—	—
1818	—	—	1863	1878	1893	1908
1819	—	—	1864	—	1894	1909
1820	1835	1850	1865	—	1895	1910
1821	1836	—	1866	—	1896	

The following table demonstrates that the disease has broken out at all the principal seaports and in some of the inland towns of British, French, German and Portuguese Colonies. It has penetrated close up to, or even into, the Soudan. It follows in Africa as in other countries, steamship routes, rivers, railroads and caravans. I think you will agree that it has been far more prevalent, and far more widespread, than is usually supposed.

Freetown	Elmina
Bathurst	Quittah
Conakry	Salt Pond
St. Louis	Accra
Dakar	Axim
Island of Gorée	Grand Bassam
Bakel, Upper Senegal	Klien Popo
Kayes & Dioubeba, Senegal	Lomé
Bamaku, Upper Senegal	Anecko
Ascension	Gran Popo
Boa Vista	Porto Novo
Cape Coast Castle	Kotonou
Secondee	Lagos
Saw Mills	Calabar

From whence did the outbreak of 1910 in Freetown and on the Gold Coast come?

I have carefully analysed a vast amount of evidence, based upon accurate records, clinical and historical, written by men of admitted ability and experience in yellow fever, which proves that this disease has been of far more frequent occurrence than is usually supposed on the West Coast. That, in fact, it has appeared annually over a very large number of years practically, as my figures show, for the last one hundred years.

A few gaps of a few years have occurred, such as between 1852 and 1858, 1868 and 1872, 1873 and 1878, 1878 and 1883, 1884 and 1890.

But from 1890 to the present date I am of opinion, from the data which I have examined, that there is an unbroken line.

During the whole hundred years there is no large interval which would make it reasonable to suppose that yellow fever had completely died out on the coast.

In my opinion this evidence is so strong that we are obliged to assume that the disease is endemic upon the West African coast for at least the last hundred years.

The question, therefore, now arises, by whom has the virus been kept up?

In the first place, we know positively that the transmitting agent, the stegomyia, is present in overwhelming quantity. It only remains to prove how a continuous source of infection has been maintained.

To those who would adopt the theory of importation, it would mean repeated importation from, say, the West Indies, Central and Southern America; the view adopted by the most recent English, French and German investigators in yellow fever is that the disease exists amongst the natives in a mild form, in other words, that it is endemic.

A little consideration will show that *the whites have not been* the source of the continuous infection, for the reason that the total number of whites on the whole West African coast has never been large enough to keep up the virus continuously; the whites are in the very small minority. We know, moreover, that the blacks can have yellow fever.

Therefore, precisely as in the case of the sister disease, malaria, the continuous or endemic source of infection is, in my opinion, the comparatively dense native population of the West Coast.

The evidence which I have examined also conclusively points both in English, French and German colonies to a vast amount of *mistaken*

diagnosis. Yellow fever was not suspected in its mild form; it never is, and it was only discovered when fatal or exceptional cases made their appearance, and, as my evidence shows, these cases were as often as not mistaken for other diseases. These same mistakes in diagnosis have occurred over and over again in yellow fever countries, especially in the commencement, notably in Cuba, where the disease was not diagnosed unless black vomit was present.

I, therefore, conclude from the evidence that a comparatively large number of deaths and mild cases have occurred from yellow fever in the past, which have been attributed to malaria, chiefly in the form known as "bilious remittent fever." Most authorities upon yellow fever are agreed that in a very large number of instances "bilious remittent fever" is another name for yellow fever.

It must be recollected moreover that it is only in this year that effective sulphur fumigation of the infected stegomyia has been attempted on the West Coast after outbreaks.

Therefore infected stegomyia were left in the past to live on and to carry infection over into a succeeding year, in those places where it was not already endemic.

Finally :—

1. The historical record of outbreaks and sporadic cases, as recorded above ;

2. Mistaken diagnosis ; and

3. The absence of any destruction of infected stegomyia in the past, is evidence overwhelmingly in favour of the disease being endemic on the coast, and of its having been repeatedly mistaken for other diseases or entirely overlooked.

III.—DIAGNOSIS OF YELLOW FEVER.

On looking over the history of yellow fever in any country, it becomes clearly obvious that great confusion has arisen over what should or should not be called yellow fever. Countries where yellow fever is long established in its endemic form, have often argued that yellow fever is only a disease of the new comer, and that as far as the residents are concerned, they do not get it, at least in the severe form. So it came about that countries like Cuba would not diagnose yellow fever unless black vomit was present. It other words it

became customary in the tropical world not to diagnose yellow fever unless the exceptional and severe symptoms were present. The mild commoner forms passed under a variety of names as we shall see later. They were essentially of the nature of an acclimatising fever; peculiar to the place, not imported; probably miasmastic, and produced by the altered conditions in which the new arrival found himself. This fever was looked upon as the necessary tax for coming to a tropical country. Those who got it became "salted" and "old coasters," and were those who subsequently openly ridiculed any idea that yellow fever could possibly be present amongst them. From time to time, however, epidemics did occur which could not be concealed, and the narratives of the survivors of the terrible symptoms fostered an exaggerated idea of the very deadly and horrible nature of yellow fever. Yellow fever soon became to be regarded as an awful and mysterious disease which paralysed enterprise and commerce. As a natural result of this there arose stricter quarantine regulations, and as a consequence of the stricter supervision, there arose a natural disinclination amongst merchants and others to declare yellow fever if they possibly could avoid doing so.

This reluctance has been universal and exists at the present day. There appears, as it were, to have been a universal conspiracy to deny the presence of yellow fever, first from the point of view that it was of no account, only attacking the new arrival, secondly, from the fact that quarantine authorities were insisting on rigid regulations; and, thirdly, that as its name caused such a panic amongst the public, the better and more business-like policy was to say nothing about the disease, either to deny its existence or return it as some other disease, or make light of it. I am convinced from many personal investigations and from the careful analysis of innumerable outbreaks of yellow fever in different parts of the world that this attitude has prevailed.

The evidence furnished to-day from West Africa shows that it certainly exists there.

Looking at yellow fever in the light of modern discoveries, the dread of declaring the existence of this disease appears foolish in the extreme; as one of the greatest authorities on yellow fever has said, there ought not to be any more hesitation about its notification than about a case of measles. The abnormal fear produced by its diagnosis is only the result of ignorance. To-day the means of controlling and stamping out this

disease are if anything more easy of execution than in the case of measles.

To return to the question of the varieties of yellow fever, there is no doubt that the young medical officer has experienced great difficulty in deciding what is and what is not yellow fever. The same difficulty crops up in every disease. Were we in a position to diagnose early the very mild forms of typhoid, scarlet fever, diphtheria, etc., the prevalence of these diseases in Europe to-day would be far different. In the majority of cases a diagnosis is not made until the well marked severe, or as they are termed "classical" signs of the disease in question have declared themselves. So it is with yellow fever: until the dreaded black vomit appears, physicians have hesitated to diagnose it; and, in the meantime, as with scarlet and other infectious fevers, the harm has been done and the disease has spread.

III.—VARIETIES OF YELLOW FEVER.

It therefore becomes a matter of the greatest importance to review our knowledge of the varieties and the symptoms of yellow fever.

It is a golden rule in analysing any disease to direct, where possible, our attention in the first instance to direct experiment or direct observation. Unfortunately a large number of tropical diseases are still based upon a most hazy and inaccurate pathological foundation. In yellow fever, however, thanks to the experimental work of FINLAY, REED, CARROLL and the other American, French, German, and Brazilian observers, we have a very definite foundation to build upon.

1. *Experimental yellow fever.*—The reason why the Reed Commission did not hesitate to make direct stegomyia inoculations, was because, as they state, FINLAY had shown as the result of 90 inoculations, that little danger resulted from the bites of infected stegomyia. In other words, FINLAY found that in 18 per cent. of his inoculation experiments there resulted *a very benign yellow fever!* The American Commission made a series of very carefully recorded observations, and in 12 cases were able to produce yellow fever, also in its milder type. The following are some typical examples:—

Case V.—Infected January 19th. On the 23rd, took to bed with feeling of lassitude and headache, temperature 99·2° F., pulse 78. Later in the day these symptoms increased, and were severe headache, chill, eyes and face flushed, temperature 100° F., pulse 104; still later, headache increased with marked backache, vomited once, temperature 103·6° F., pulse 110.

At the end of 42 hours albumen appeared in the urine. The scleræ became jaundiced. On the second day and by the fourth day this had extended to other parts of the body. The symptoms did not increase and on the seventh day the temperature was normal.

Case VI.—C. W., American, non-immune, aged 27, with his full consent was at 9.30 a.m., January 31st, bitten by two of the three mosquitoes that had been applied to the foregoing Case V. The interval that had elapsed since their contamination was, therefore, 51 days. The subject remained well until 12 o'clock noon, February 3rd, when he complained of heaviness in his legs and some supra-orbital pain. His temperature at this hour was 99° F. and pulse 70. At 1.30 p.m. it had risen to 100° F., and at 5 p.m. to 100·6° F., and pulse to 84. The primary rise of fever, which was somewhat fluctuating in character, did not reach its height until at the end of 24 hours, noon, February 4th, when the temperature was 102·4° F. and the pulse 92. The facies was now suggestive of yellow fever. Remission occurred at the end of 45 hours and lasted for about one day. The secondary rise was slight in character, the temperature falling to normal on the morning of the sixth day. The case was very mild in character. Albumen appeared at the end of 75 hours (beginning of fourth day); it never amounted to more than a distinct trace and disappeared on the eighth day. There was no ocular jaundice, and although the gums were injected and swollen, there was no hæmorrhage at any time. The patient perspired freely throughout the attack. Convalescence was rapid. The subject had been in quarantine for the period of six days prior to inoculation.

Case VII.—J. H., American, non-immune, aged 26, with his full consent was bitten at 11 a.m., February 6th, 1901, by the same two mosquitoes that had six days previously bitten Case VI. Fifty-seven days had therefore elapsed since the insects had been contaminated by biting a case of yellow fever He remained well until 12 o'clock noon, February 9th, when he experienced slight chilly sensations, accompanied by yawning. At this hour his temperature was 99° F. and pulse 72. At 3 p.m. temperature 98·8° F. pulse 72. Says that he feels 'out of sorts' but has no headache. At 5.30 p.m. his temperature was 100·6° F., pulse 78. He was not seen until 7.30 p.m., when he complained of backache and severe general headache, more intense through the frontal region. Eyes much injected, photophobia very marked, face flushed. He was stretching and yawning constantly, complained of nausea, and vomited a small quantity of partially digested food. The height of the primary fever was reached at 3 p.m., February 10th, *i.e.*, 22 hours after the commencement of the attack, when the temperature was 102·8° F. and the pulse 98. Remission of the fever to 99·4° F. and the pulse to 74, occurred at 6 a.m., February 11th, making the duration of the primary paroxysm 36 hours. Twenty-four hours after the remission had occurred, the temperature had risen to 102·4° F. with a pulse of 70. The fever continued to steadily increase until midnight of February 12th, when a temperature of 105° F. was recorded, with a pulse of 90. The subsequent course was that of a case of severe yellow fever. Slight oozing of blood from the gums occurred as early as the third day. Ocular jaundice, beginning on the third day, became later very distinct, and was associated with general jaundice. Albumen, however, did not appear until the sixth day. The fever subsided on the ninth day, and was followed by a slow convalescence. The subject had been in strict quarantine for a period of 78 days prior to inoculation.

Case VIII.—C. S., American, non-immune, aged 28, with his full consent, was bitten at 2 p.m., February 7th, 1901, by three mosquitoes that had been contaminated sixteen days previously by biting a fatal case of yellow fever on the second day of the disease. The subject remained well until the early afternoon of the third day, Feb. 10th. He ate his dinner at noon with good appetite. At 1 p.m., his temperature was 100° F., pulse 90. Frontal headache was now complained of. This became rapidly more marked and pains in the limbs were also present. Three hours later, 4 p.m., the fever had risen only one-fifth of a degree, but at 6 p.m. it had reached 103° F. and

pulse 96. At this hour he had severe headache and backache with general pains over the body. The facial expression was now characteristic. Eyes brightly injected, photophobia well marked, and face decidedly congested. At 9 p.m., temperature was 103·2° F. and pulse 112. The skin was moist. Remission occurred at 6 a.m., Feb. 12th, 41 hours after the commencement of the febrile paroxysm. The secondary fever, which was moderate in character, lasted four days, the fever subsiding on the seventh day. Ocular jaundice was slight. A few hyaline casts were present on the third day, and subsequent days, but no albumen was found until 1 p.m., February 18th, that is, 36 hours after the fever had subsided. It was present as a distinct trace in all specimens examined until 3 p.m., February 23rd. Convalescence was rapid.

In all these experimental cases the Cuban Board of yellow fever experts had no doubt about the nature of the disease. Yet we must all agree that the symptoms appear very mild: headache, backache, chill, lassitude, a rising temperature, the pulse not following, a trace of albumen in the urine, occasional vomiting, jaundiced scleræ, flushing, gums spongy and shewing a tendency to bleed, a remission and then a second attack. These are certainly not symptoms which a medical officer would pronounce to be those of yellow fever, unless he was looking out for the disease. The great authority, CARROLL, has stated that their experiments show that genuine yellow fever may be so mild in character that no man, no matter how extensive his experience may have been, would dare to diagnose it as such, unless he knew the disease to be prevailing at the time.

Such cases at the beginning of an outbreak would render it extremely difficult, or impossible, to trace the origin of the severer ones occurring later.

2. *Inflammatory Fever.*—In the light of precise experimental data we may then, I maintain, be prepared to find that yellow fever has masqueraded under many euphemisms as the following shows: The "inflammatory fever" of the West Indies has been long recognised as an acclimatising or seasoning fever, which newcomers were expected to get. It was another name for some local or endemic malady about which no one knew anything except that all were expected to get it, at one time or another.

The older writers* wrote much about it, and held that it did not have any strict affinity with the dreaded yellow fever. The symptoms recorded were lassitude, chill, injected eyes, flushing, fever, pains, frontal headache, lumbar pain, nausea, vomiting, epigastric tenderness, urine scanty and

* DICKINSON: Observations upon the Inflammatory Endemic incidental to strangers in the West Indies, commonly called the Yellow Fever. London. 1819.

high coloured. These symptoms might become more marked, the headache might become more violent, also the epigastric tenderness, persistent vomiting, delirium, and elevation of temperature. Later, again, marked yellowness of the skin. In still more severe attacks: persistent black vomit and hæmorrhages, tarry stools, violent delirium, coma and death. In this picture of the inflammatory fever of the West Indies you will agree there are the various types of yellow fever—the mildest to the most severe forms.

More recent writers have drawn attention to this disease, because it explained the recent outbreak, 1909, of yellow fever in Martinique and Gaudeloupe. Most of our knowledge is due to SIMOND, AUBERT and NOC, who have been engaged on more than one yellow fever investigation, and who were in the West Indies when I was there last year on yellow fever enquiries. These investigators show that the symptoms of inflammatory fever are identical with those of experimental yellow fever. They give cases where a mild attack of inflammatory fever passes into the severe form of yellow fever. The symptoms can be so mild as to produce only an *embarras gastrique.*

The symptoms are usually slight fever, vomiting, albuminuria, jaundice on the fourth or fifth day, a remission, then a rise. The disease is not fatal, nor does it confer absolute immunity. It may occur quite independently of any case of severe yellow fever and may occur in epidemic form ; it often precedes an outbreak of malignant yellow fever, but it may not do so. They conclude that under inflammatory fever may sometimes be included either mild cases of yellow fever or cases of malaria. They also state that yellow fever is characterised more by the milder symptoms than by the malignant.

As in the case of experimental yellow fever so in inflammatory fever, the patients are as capable of giving infection to the stegomyia as virulent cases of yellow fever.

There can be no doubt, therefore, both from the evidence of old clinical investigators, as well as of modern experimental investigators, that this endemic acclimatising or seasoning fever is in reality very often genuine yellow fever, and is the source from which the stegomyia becomes infected. Like experimental yellow fever, it would be overlooked did not severe types of yellow fever occur from time to time with it.

The existence of inflammatory fever is as significant as the existence of severe yellow fever, and proves that yellow fever is endemic.

3. *Bilious Remittent Fever.*—This fever takes the place in West Africa of the inflammatory fever of the West Indies. It is a very old name, and typified the "acclimatising," "seasoning," or "endemic" fever of any locality which newcomers were expected to get. Volumes have been written upon it. Some of the older writers held that, just as in inflammatory fever, it could pass into severe yellow fever; others held that it was a specific entity. Others, again, that bilious remittent fever carried from one locality could burst out into the malignant yellow fever on arrival in another country. All these doubts and theories pointed to the fact that the older clinicians were observing one and the same disease, but of different types of severity. From investigations of careful records of outbreaks of yellow fever, there can be no other conclusion than that a very large number of cases of genuine yellow fever have been classed as "bilious remittent fever." Bilious remittent fever may also, just as inflammatory fever, include a proportion of malaria cases, but it undoubtedly has very frequently been used as another name for yellow fever. For example, Colonel Birt, in a recent paper published in the *Journal of the Royal Army Medical Corps*, refers to the outbreak of yellow fever in Malta, and how in 1881 there are entered sixty-nine cases of bilious remittent fever with six deaths. He points out that primary malaria is exceedingly rare in Malta, and is limited to one remote valley. He concludes that the term "bilious remittent" was only a euphemism for yellow fever. Bilious remittent was at one time like yellow fever common in Barbados, where there are no anophelines. Carroll refers also to this disease in the following decisive terms:—"We know, as a matter of fact, that here in the United States yellow fever has been time and time again called 'bilious remittent fever,' until the occurrence of a number of fatal cases with black vomit proclaimed the true nature of the disease. There is good reason to believe that, with the approaching complete control of yellow fever, the old-time bilious remittent fever will become much less frequent in the United States."

In an official report, drawn up for the Marine Hospital Service in the United States in 1898, the following statement occurs:—"The bilious remittent fever, that in our old text-books of medicine occupied such a

conspicuous place in tables of differential diagnosis with yellow fever, has practically disappeared from the Southern Sea border, since yellow fever ceased to be an endemic there. It was, in fact, the yellow fever of the natives and of places in the interior." This is a most significant statement, for it proves that bilious remittent fever has in places diminished or disappeared *pari passu* with yellow fever. Of course, the malarial forms of bilious remittent may continue to exist. But pipe-borne water supplies appear to have affected bilious remittent fever precisely as they have done yellow fever.

Let us now examine bilious remittent fever in the light of old and recent observations, more especially as it affects West Africa. One of the best accounts of this disease will be found in the first volume of the *British Medical Journal*, then the *Provincial Medical Journal, 1842*, by C. J. BURTON, Surgeon to the Royal African Corps.

He classifies the fevers of West Africa into bilious remittent fever, simple bilious fever, intermittent fevers, yellow fever.

With regard to yellow fever, he states that it generally appears in epidemic form every seven years in Sierra Leone. He mentions that its origin had given rise to much debate; some regarded it as imported, but the majority were of opinion that it had a local origin. BURTON believed in the local origin, and the great majority of writers, both before and after BURTON'S time, have held a similar opinion. He then goes on to state that "yellow fever has been considered by many as an aggravated form of remittent fever, and the idea is supported by the fact that all the symptoms are similar, only much more severe in the former. This error, however—for error it must be considered—originates, I am led to think, in mistaking the severer types of bilious remittent for yellow fever, and it must still be considered that the latter is a specific disease; one, in fact, *sui generis*." BURTON describes how bilious remittent fever, or "country fever," proves fatal every year to a large number of people; and how every year it appears in an endemic form, it can never be said to be wholly absent, and not unfrequently *appears in the dry season*.

BURTON describes the symptoms as follows:—"The symptoms are extremely varied, and scarcely ever appear in the same order in any two persons attacked. There is, however, a sufficiently well-marked similarity in all cases to lead, after a little experience, to an immediate and correct diagnosis."

"Sometimes, indeed in the majority of cases, the disease is ushered in by a sudden chill, generally referred by the patient to the small of the back, in some cases extending along the whole course of the spine, and likened to the trickling of cold water down the back. The cold feeling just described sometimes amounts to a complete rigor, but in the greater number of cases the chill is only momentary, and at other times no sensation of cold whatever is felt. The rigor, or momentary feeling of cold, as the case may be, is soon followed by reaction; at first, alternate flushes of heat and cold are perceived; by degrees the skin becomes either moderately or excessively hot; if the latter takes place, the patient feels as if surrounded by furnaces, and tosses about in the most restless manner. There is usually a dizzy feel of the head from the beginning; but as the hot fit advances, headache, sometimes of the most excruciating description sets in; there is usually a feeling of stupor during the afternoon, at which time the fever is at its height."

"The disease sometimes commences with vomiting, generally of bile, either in a moderate or excessive degree; but there is at other times merely a degree of nausea; and in some cases neither of these symptoms appears. At the beginning there is usually a ringing sound heard in the ears; the eyes are dull, and more or less sunken; the conjunctivæ are red and appear injected; the countenance is expressive of suffering and anxiety; the tongue puts on different appearances in most cases, and usually forms a good indication as to whether the disease is likely to prove severe or otherwise; if the latter, it is at the beginning of the fever white in the middle, and as the disease advances it becomes coated with a brown fur; but if the tongue is red, or coated with a white tenacious fur, with extremely red edges, then a severe and dangerous attack may be apprehended. Sometimes, during the progress of a dangerous attack, the tongue becomes dry and black. No symptoms portending worse consequences than this can show itself; but, happily, even under such discouraging circumstances, the case is not to be considered as necessarily fatal. There is usually an unpleasant feeling at the pit of the stomach; sometimes a burning sensation is complained of; in other cases it amounts to a feeling of pain, especially on pressure. The bowels are nearly always extremely costive, though the disease is now and then ushered in by bilious dejections. The urine is high coloured and scanty; in some rare cases there is frequent and painful

micturition, evidently depending on irritation or subacute inflammation of the mucous membrane of the bladder. The thirst is in most cases excessive; some patients will swallow gallons of fluid in the course of the twenty-four hours. Pains in the different muscles are often felt; but few cases are unaccompanied by great pain in the joints, the knees appearing to suffer most. Sometimes delirium appears in a few hours after the commencement of the attack. There are, however, few patients who pass through the disease without being more or less affected with this disagreeable symptom at some period of the fever, especially during the night."

"When it occurs only at night, it is a combination of delirium and unpleasant dreams, extremely annoying and fatiguing to the sufferer. If delirium appears during the day it is fierce, and attended with much greater excitement and mental aberration. In some patients the disease sets in suddenly and violently; sometimes the actual period of invasion cannot be ascertained within any degree of precision. In these latter cases the person feels for a day or two a certain degree of languor and lassitude, with feebleness of the lower extremities especially from the knees downwards, and partial loss of appetite. These symptoms become gradually increased, and finally so aggravated that the patient is obliged to keep his bed, and a regular attack of fever sets in."

In the beginning of the disease there appears very little alteration in the pulse; but when the febrile symptoms become fully developed it is either quick, full, and bounding, or exceedingly quick and hard; at other times it is only accelerated and full. The state of the pulse, however, depends more on the constitution and temperament of the person attacked than on the comparative severity or mildness of the case. The symptoms described may appear either in a mild form at first, and afterwards become partially or greatly aggravated, or they may from the first set in with the utmost severity. In some cases the irritability of the stomach increases to such a degree that matter strongly resembling the "black vomit" in yellow fever is thrown up, and frequently a yellow tinge of the skin, chiefly about the neck and chest, is perceived. It is necessary, therefore, to bear in mind that there are two ways in which this fever commences. The first is where it sets in with violent symptoms, accompanied by great heat of

skin, headache, delirium, etc. This may be described as the tangible form, and is certainly the least dangerous, if promptly and energetically treated. The second when the symptoms are all more or less obscure, there is but slight heat of the skin; when the hand is applied there is a feeling of dryness rather than heat. There is a constant dull pain in the head, scarcely amounting to headache; no delirium is present, but there is slight confusion of ideas during the night. The tongue is usually, in this form of the fever, red, or the edges are very red and the middle of the tongue is white; in fact, all the symptoms of fever are present, but in so low and obscure a form that the nature of the disease might be mistaken until too late. This is decidedly the most dangerous kind of fever, and may be called the obscure form.

With regard to the remissions, he states that a remission may occur or the type may be continuous. If it does occur, it appears generally on the third day, when the diminution of symptoms is such that the patient feels well enough to rise from bed.

BURTON lays stress on a very significant fact in connection with bilious remittent fever, namely, the generally accepted doctrine with which he agrees, that one attack of the fever confers immunity against a second attack. This is, of course, of great importance, because it points to a disease other than malaria, and is in harmony with our knowledge of experimental yellow fever, inflammatory fever and yellow fever in all its types.

BURTON then describes "simple bilious fever."

He first mentions that some might regard it as a milder form of the bilious remittent; he, however, was inclined to regard it as a specific entity. The disease is ushered in with headache, vomiting of bile, fever; it attacks suddenly, usually with vomiting, retching and nausea. In the majority of cases, pains in the head, sometimes very severe. Delirium is not common, pulse quick and full. Tongue coated, urine high coloured, scleræ slightly jaundiced.

Returning to bilious remittent fever, BURTON states that it is the endemic remittent fever of all tropical countries occasionally occurring in northern climates during an *unusually* hot autumn. He describes it as met with in the West Indies, in a severe form, in the East Indies in a mild form, as causing much mortality in British Guiana and British Honduras, and in the southern parts of the United States, and has

appeared in an extremely severe form in some of the Spanish Provinces. In other words, with the single exception of the East Indies, bilious remittent fever has been common wherever yellow fever has been present. He then states that of all countries it is most severe in West Africa, that a more treacherous fever cannot be conceived. He then appends the following tables, drawn up by FERGUSON, for the troops of the Royal African Corps for 1825 :—

Stations.	Strength.	Treated.	Died.	Deaths to Cases treated.	Deaths to Strength.
First Quarter.					
Sierra Leone ...	188	—	—	—	—
Gambia	—	—	—	—	—
Isles de Los ...	108	—	—	—	—
Second Quarter.					
Sierra Leone ...	289	46	13	1 to 3.61	1 to 22.2
Gambia	140	3	—	—	—
Isles de Los ...	106	5	2	1 to 2.5	1 to 53
Third Quarter.					
Sierra Leone ...	585	386	161	1 to 2.39	1 to 3.63
Gambia	108	92	74	1 to 1.24	1 to 1.45
Isles de Los ...	103	99	23	1 to 4.3	1 to 4.47
Fourth Quarter.					
Sierra Leone ...	522	146	37	1 to 3.94	1 to 14.1
Gambia	112	89	60	1 to 1.47	1 to 1.86
Isles de Los ...	75	120	7	1 to 17.1	1 to 10.7

BURTON states: "From the above table, drawn up by Surgeon FERGUSON of the Royal African Corps, it will appear that in a regiment never exceeding 796 men, there were treated in one year 986 cases of fever, and of these 377 died, giving a mortality of nearly one half the total strength from one disease alone. Although authorities might be multiplied in proof of the extremely dangerous and fatal nature of the fever in

question, one extract only, from the report of a medical officer of talent, and high standing in the service, will be given. These reports were made about sixteen years ago, but recent events upon the coast fully prove that the climate remains the same, and that Europeans cannot approach these regions of malaria without exposing themselves to the greatest danger. "The arrival of twelve sergeants," says this officer, "forms a striking feature in the events now under consideration, and affords a gloomy illustration of this deleterious climate. They were selected from the detachments at the Isle of Wight; men of good character; their conduct, at least on this coast, was as exemplary as could be expected from men of their class in society. Some irregularities, of course, occurred; but had they been sinners above all sinners we could not have expected so desolating a retribution. The whole of them were attacked with fever, and within a few months after their arrival eight paid the debt of nature, and only one at present (the sergeant-major) appears fit for duty. The constitutions of the other three are in such a shattered state that plainly indicates an advanced stage of visceral disease. The sickness and mortality amongst their wives and children are nearly in the same proportion." This affords a very fair specimen of what may be expected from the effects of the "bilious remittent fever" of Western Africa, without taking into consideration the many other diseases which attack the European residents. In fact, this is nothing but the process of "seasoning" through which every stranger must pass in a few months after his arrival in the country."

Impartially reviewing BURTON'S descriptions of bilious remittent fever in the light of experimental yellow fever and inflammatory fever, and bearing in mind that BURTON'S observations were long before the Stegomyia doctrine or the discovery of the parasite in malaria, one is forced to the conclusion that by this name is described some of the typical types of yellow fever. In order, however, to still further support my view that under the term "bilious remittent fever," yellow fever is very frequently included, I induce the following further evidence:—

BOYLE, a well-known Naval authority on tropical fevers, and also the Colonial Surgeon of Freetown, devotes a whole chapter to climatorial bilious remittent fever, by which he meant a fever which broke out on ships, of the type of bilious remittent fever. This is very significant because it shows the great difficulty which BOYLE had in conceiving why

bilious remittent fever should occur spontaneously on board a ship, the crews of which had not been ashore for any length of time. Naturally it is the old story of yellow fever over again, and shews the difficulty of explaining the occurrence of yellow fever on ships, and likewise that of bilious remittent fever. It would be quite impossible to explain either, unless on the doctrine of the presence of the Stegomyia. But let us analyse some of BOYLE'S cases, abstracted from his journal on board the H.M.S. "Cyrene" in 1822-3.

Case I.—James Jones, seaman, *æt* 35, had been ashore fourteen days previous to sailing; taken ill with headache and pains in body, fever, pulse 90: later delirium. Ended fatally.

Case II.—John Atherton, at sea. Symptoms: Intense headache, lassitude, thirst, fever, pains in body, nausea. Recovery

Case III.—Samuel Simmonds, at sea. Severe pain in head, back and loins, weakness, griping and purging fever. Next day pulse reduced in frequency. Recovery.

Case IV.—William Hunter, on board a schooner at Sierra Leone. Symptoms: Intense pain in head, back and loins, fever, pulse 130, vomiting, great thirst. Later, severe vomiting and epigastric tenderness developed, stupidity, pulse and temperature reduced. Post mortem: the stomach was deeply congested towards pylorus.

Amongst the numerous cases of bilious remittent fever which developed on the "Cyrene" there occurred one case of fatal yellow fever, characterised by black vomit and jaundice. Surely this case gives the clue to the origin of the cases of so-called climatorial bilious remittent fever which occurred on board. Very many other cases are given together with the post mortem findings and there cannot be a doubt that some were genuine malignant yellow fever cases and other milder types. These cases occurring side by side with fatal cases, which were either diagnosed at the time as yellow fever, or which, from the description of the post-mortems, I have no doubt were yellow fever, is unmistakable evidence of the yellow fever nature of the climatorial bilious remittent fever.

BOYLE then devotes a chapter to his *local bilious remittent fever* and here again, it is abundantly evident that he is dealing with one of the common milder types of yellow fever.

He describes it as the acclimatising fever of the locality which every new comer had to get. He lays particular stress upon the fact of the exclusive predisposition of the English and northern races to the disease. The symptoms are chill, malaise, pain in head and body, fever, bilious vomiting, congested eyes, yellowness of skin and eyes. In some cases

tarry stools and dark vomit, delirium. He then notes how sporadic cases of yellow fever break out side by side and he calls them aggravated cases of the endemic bilious remittent. So here again it is clear that we are dealing with one and the same disease, viz., yellow fever.

BOYLE gives pages of description to this fever, and it is clear that it passes insensibly into yellow fever.

The evidence of Surgeons BARRY, LAWSON, FERGUSSON and TIDLIE, BURNETT and many others, given either in books or in MS. records, all points to the one conclusion, that cases of bilious remittent fever and yellow fever occurred together. When the fever appeared in epidemic form or with black vomit it was yellow fever. otherwise climatorial or endemic remittent fever. Simply a repetition of the picture of "inflammatory fever" in the West Indies; all degrees of severity from *embarras gastrique* to yellow fever. All authorities were agreed that the native (comparatively speaking) very rarely suffered from the severe black vomit type, and they also observed that the bilious remittent gave a certain degree of immunity against yellow fever. Now to turn to the recent evidence furnished by the 1910 epidemics.

In the first place, at the commencement of the outbreak of yellow fever in Secondee, came a stout denial on the part of the Press and some of the medical men that yellow fever existed, or even could exist, on the Coast. They seriously declared that the disease was only the malignant form of bilious remittent fever with which they were well acquainted, and which usually attacked persons towards the rainy season. This statement is quite true, if the bilious remittent fever is regarded as true yellow fever and has, as the newspapers state, been no doubt present every year.

I have carefully examined a large number of case books and other records at Secondee, Accra, Cape Coast, Salt Pond, Elmina, Axim, Quittah, Lagos and Freetown, and I have found that amongst the cases returned as yellow fever, there were others diagnosed as bilious remittent fever or simple remittent fever.

At Lagos I was particularly struck by the very large number of remittent cases. Of course a certain proportion of these are in all probability malarial, but with equal certainty we can say that a proportion are mild cases of yellow fever. It is beyond dispute then that during outbreaks of fever with well-marked black vomit there have

occurred in the present year and in past years numerous cases of "remittent" and "bilious remittent fever." This is not a new observation peculiar to the West Coast, the same fact has been observed wherever yellow fever has been present.

I will now give an example of one of the cases of yellow fever which occurred in Secondee during the recent outbreak, and which was regarded as bilious remittent fever by one medical man:—

Mr. D. had been in the Colony a few months on this occasion, but had been on the coast before. Taken suddenly ill May 19th. May 21st, vomited, severe headache, temperature 101°, jaundice, no albuminuria. May 22nd, temperature 102°, jaundice deepened, conjunctivæ congested, urine scanty, no albumen. May 23rd, temperature 102°, albumen present in the urine, vomited material like beef-tea dregs. May 24th, temperature 103·4°, urine scanty, very albuminous, jaundice, pulse 70, vomit speckled with black spots. May 25th, temperature 101°, restless, urine scanty. May 26th, temperature 98·8°, pulse 80, urine loaded with albumen scanty, vomited, petechiæ present on body, jaundice deep, hiccough. May 27th, temperature 98·8°, pulse feeble, urine suppressed, hæmorrhage from mouth, death,

Post Mortem.—Surface of body yellow, petechiæ, liver yellow and congested, recalling the appearance of nutmeg liver, neck of the gall bladder deeply congested. Stomach contained a copious quantity of black grumous material, also the intestines, "stomach and intestines stained dark." There can be no doubt about this case belonging to the severe type of yellow fever. It was one of the several that occurred one after the other, but, nevertheless, a native medical man comes forward and states as follows:—"The case of the late Mr. D., whose autopsy I witnessed yesterday, was one of *malignant bilious remittent fever* and not one of the true pestilential yellow fever," . . . . therefore, all the preceding cases which were on all fours with Mr. D.'s case, were malignant bilious remittent fever and not yellow fever"!!

This medical man is not an exception; he is an example of many who go about encouraging the belief that yellow fever does not exist. It is of course clearly obvious that this attitude proves that malignant bilious remittent fever of West Africa is in a *very large number* of cases, genuine yellow fever in its various degrees. It is now our duty to drive this home, and to treat all cases of bilious remittent fever as cases of yellow fever, unless a very clear proof is furnished that they are due to the malarial parasite. I feel confident that if this view is adopted and anti-stegomyia measures undertaken, that not only will yellow fever disappear, but that bilious remittent will also largely vanish out of Africa.

V.—SYMPTOMATOLOGY OF YELLOW FEVER.

Under experimental yellow fever, inflammatory fever and bilious remittent fever I have already described very many of the symptoms of yellow fever. The point to bear in mind is the wide range of the symptoms from an *embarras gastrique* and feeling of lassitude, to

the severe symptoms culminating in black vomit and suppression of urine. The symptoms usually commence with a chill and feeling of lassitude, headache, lumbar and joint pains, which become more intense.

The temperature is above normal, and continues to rise for 24 to 48 hours, reaching 104·5° F. or even higher, but not usually. The temperature may continue for four or five days more with slight variations, or as often happens, there is a remission or the temperature curve may present several remissions. The pulse is that of any fever, it is quickened 100 to begin with, but as the fever progresses the pulse slows down to 90, 80, 70, or 65, whilst the temperature may stand at 100°, 102°, or 104° F.

This want of accord between pulse and temperature, although by no means constant, is exceedingly characteristic, and serves to stamp the disease as very suspicious of yellow fever. It is known as Faget's sign. The tongue is coated in the centre, and the edges and tip are red. The gums are sore and often present a fine red line of congestion where the lead line is usually found. Nausea and vomiting may commence very early, the vomit consisting at first of food products, then thin mucus, or bile-stained mucus, whilst on the fourth or fifth day, or even earlier, small black specks are observed in the vomit. Subsequently the typical black vomit sets in, or blood may be vomited. The stools are now often tarry. The urine is a febrile one to commence with, then after the end of 48 hours a trace of albumen is usually found, this increases and at the same time the urine diminishes in quantity and finally may end in total suppression.

Yellow discolouration usually sets in about the third day, and is first noticed in the scleræ, then it spreads to the rest of the body. It is characteristic to observe the deepening of the colour as the disease progresses. Or the yellow colour may not appear till after death, or during the death agony.

When once seen this appearance can never be forgotten. Congestion of the eyes and of the upper part of the upper part of the body is often seen, and petechiæ may appear. Hæmorrhages are common from the nose, mouth and other orifices. The remission, referred to above, may last for several hours, and very often gives a false sense of security; I have noticed it many times, and it is a characteristic and suspicious

sign. The patient feels well, wants to get up and take a meal. It is succeeded by a relapse and the temperature rises. This is sometimes known as the second paroxysm or the reactionary fever.

The following history sums up a case as it occurred in the 1847 epidemic in Freetown. It is very graphic and accurate:—

John Ogden, Marine of H.M.S. "Growler," was attacked with remittent fever on the 15th March, 1847, and on examination was found to be labouring under the following symptoms on the 16th . . . . . Considerable frontal headache, flushed countenance, skin hot and dry, tongue covered with a yellow fur in the centre, being slightly red and dry in the tip, and edges, constant nausea, occasionally vomiting a bilious-looking fluid, accompanied by epigastric tenderness; pulse quick and full, urine scanty and turbid, depositing a latentious sediment on cooling—bowels confined, occasional attacks of shivering, accompanied by pain along the spine. A sample of calomel was administered immediately, followed in three hours afterwards by a solution of sulphate of magnesia in peppermint water, a sinapism was applied to the epigastrium, and the body sponged with lime juice and water. In the evening a remission took place, and he felt himself much better in every respect; the bowels having been freely acted on by the medicines producing several stools, the evacuations being fœtid and of a dark brown color, three grains of calomel and a quarter-grain of opium were given every second hour until the system was brought under the influence of the mercury, which occurred on the 18th. Three days after the accession of the disease the symptoms daily decreased in severity from this period, and on the morning of the 21st he stated that he had slept well during the night, that he felt quite well, and wished for something to eat. He was allowed two ounces of arrowroot for dinner, which he enjoyed very much, and at the evening visit was quite free from any febrile symptom. About 11 o'clock the same night he suddenly screamed in his sleep, started out of bed, and attempted to jump out of the window. On visiting him immediately I found him labouring under great mental excitement, his face flushed, his eyes brilliant, skin dry and burning, with slight tremor of the hands; pulse rapid, but soft and easily compressed, he spoke incoherently, and had to be restrained in bed. He vomited a considerable quantity of a dark coloured fluid resembling *coffee grounds*. His head was immediately sheared, and a large blister applied across the vertex, from ear to ear. A drachm of the ung. hydr. was rubbed into the groins every second hour, three grains of calomel and a quarter of a grain of opium were administered every hour. Notwithstanding the active remedial measures employed symptoms of coma came on in a few hours afterwards, and he died at nine o'clock on the following night (the 22nd).

POST MORTEM APPEARANCES

Cranium.—The sinuses and central veins slightly congested. Membranes of brain perfectly healthy. There was about two teaspoonfuls of serum in the lateral ventricles.

Thorax.—Lungs and heart healthy.

Abdomen.—Stomach was found half filled with a dark coloured fluid, similar in appearance to that vomited, having some resemblance to the "black vomit," but on minutely examining it, it was found to consist of *shreds of lymph*, mixed with the secretions of the stomach and the medicine administered. The membrane covering the cardiac extremity was easily separated from the other coats, and presented various patches [illegible]. The remainder of the abdominal viscera were perfectly healthy.

VI.—OBSTACLES TO DIAGNOSIS AND NOTIFICATION FEAR.

It seems very strange that there should be obstacles in the way of the diagnosis of yellow fever; but such is the case. In all the outbreaks which I have had to investigate I have encountered them, and writers upon yellow fever have, time and time again, drawn attention to this point.

I encountered it in a particularly gross form in Barbados last year (1909), and I was the means of drawing attention to it, and I venture to hope that I may have been able to stop it. It arises primarily from the press and mercantile community; and these agencies, in their turn, slowly but surely influence those in authority as well as the younger members of the medical profession. There is absolute notification fear. I have known personally medical men who have been persecuted because they dared to notify yellow fever. I have examined the correspondence in other cases where the medical officer making the diagnosis has been promptly sat upon, and where in consequence the opinion, he secretly held, had been abandoned in deference to the view that it was better not to diagnose yellow fever. This year, in Africa, the press called into serious question the diagnosis of the reputable medical authorities of the colony, and stoutly upheld and liberally praised those who diagnosed "bilious-remittent fever." Press vilification is common all over the world, and it therefore becomes all the more incumbent upon those who are in administrative authority to uphold the decision of their medical officers. I will give three examples of notification fear in West Africa.

In 1884, a Doctor DAVIES, practising in Freetown, reported a case of yellow fever. Upon receipt of this report the Acting P.M.O. sent him a letter asking him the grounds upon which he made the diagnosis, and if "he considered the case of yellow fever which he had had in his practice to be contagious or not?" In a letter to the Governor the Acting P.M.O. stated in his opinion that Dr. DAVIES had made a grievous mistake in his diagnosis of yellow fever.

In 1903, a Cape Coast doctor, Dr. BARKER, wrote the Acting P.M.O. reporting a case of yellow fever, and stating that it was the fifth case. The medical man who made this diagnosis was Dr. ROME HALL. The Acting P.M.O. wrote as follows to the Colonial Secretary: "I regret to say that I am obliged to take the alarmist statement made by Dr. ROME HALL with a large amount of doubt."

In 1910 there is an improvement as regards the majority of the medical officers, who have had the courage of their convictions and did not hesitate to express them. A few held, however, that the disease was the well-known "bilious remittent fever," and the press, as we have seen, did not hesitate to openly abuse those who diagnosed yellow fever.

VII.—MISTAKEN DIAGNOSIS.

Excluding inflammatory fever and bilious remittent fever, which I have already fully discussed, and which are in a large number of instances cases of genuine yellow fever, there remains a long list of mistaken diagnoses which have been made instead of yellow fever:—

1. **Pernicious remittent fever.**
2. **African fever.**
3. **Typhoid.**
4. **Enteric fever.**
5. **Pernicious remittent fever on the borderland of yellow fever.**
6. **Non-contagious yellow fever.**
7. **Typho-malarial bilious remittent fever.**
8. **Acute delirious malignant malarial fever.**
9. **Hyperpyrexial fever.**
10. **Subacute rheumatism.**
11. **Bilious remittent fever with morbis cordis.**
12. **Pernicious fever with nephritis.**
13. **Febris malaria continua perniciosa nephritica.**
14. **Malignant jaundice.**
15. **Gastric influenza.**
16. **Epidemic jaundice, or Weils disease.**
17. **Forms of malaria.**
18. **Dengue fever.**
19. **Influenza.**
20. **Bilious fever and hæmatemesis.**
21. **Gastric ulcer.**
22. **Phosphorus poisoning.**
23. **Hepatitis or acute yellow atrophy.**
24. **Continued fever.**

The following are a few illustrations of mistaken diagnosis amongst very many:—

Salt Pond. Examination of cases from 1889 *to* 1903.

In 1895 a case occurred—diagnosed as "remittent fever"—terminating fatally. It was characterised by jaundice; temperature 105·8 F.; pulse 60; black vomit; suppression of urine: hiccough: intense headache.

There were also about this time numerous other cases with a similar diagnosis presenting very suspicious symptoms.

In 1897 yellow fever was diagnosed.

In July, 1897, a case was diagnosed as hæmoglobinuria and acute nephritis.

It was not diagnosed as yellow fever because of the absence of black vomit Faget's sign; jaundice, and hiccough were, however, present, and hæmorrhage from gums as well as vomiting of dark bilious fluid is mentioned.

So, considering there were other cases in that year, it is not improbable that this was also a case of yellow fever.

In June, 1896, a case occurred, ending fatally, diagnosed as "'acute' delirious malignant malarial fever.'"

The patient was so maniacal that he had to be handcuffed; temperature 105·4 F. and rose to 107·2 F.; skin jaundiced, head and shoulders intensely congested, nausea, vomiting, coma and death. Post mortem: Cutaneous hæmorrhages, mucous membrane of stomach intensely congested, stomach contained dark green treacly mass, liver saffron yellow, with patches of congestion. This might well have been a case of yellow fever in spite of the fact that it is stated "pigmented corpuscles were found in the blood."

In 1901 two cases were diagnosed as yellow fever; but in addition in each year there occurred cases which, when we know that there were genuine cases of yellow fever, might equally well have been cases of yellow fever. The diagnoses made were usually "bilious remittent fever."

Axim. Examination of case books 1906-10.

In 1905 the medical officer in charge of a case which occurred in that year at Axim, and which terminated fatally, is now of the opinion that the case was one of yellow fever.

In this year, 1910, a death occurred from yellow fever and was reported. The symptoms were, however, not well marked.

Later on in 1910 another fatal case occurred, which was diagnosed as "remittent fever." The chief symptoms were: temperature 104·9; pulse rate from 110-120. Temperature did not yield to large doses of quinine.

Another case was diagnosed as "pernicious remittent fever" and proved fatal.

The symptoms were: intense headache; temperature 102·8: pulse 88; delirium, and finally coma.

The doctor in charge suspected yellow fever.

CONCLUSION.

Yellow fever is endemic and common in West Africa, and has occurred in all its various forms from the *embarras gastrique* to black vomit.

There is but one remedy, and that is stegomyia destruction; and there is but one result to expect, namely, a most marked increase in the health and prosperity of West Africa.

Considering the great importance of the subject and the short time available for discussion, it was agreed to defer the discussion until the next meeting of the Society, but it will be interesting to note the following questions addressed by Sir PATRICK MANSON, and the answers given by Sir RUBERT BOYCE:—

1. Assuming Sir R. Boyce's hypothesis to be correct, [illegible] show itself in the natives [illegible]

2. Why did the native of Barbados, who for many years had enjoyed immunity from yellow fever, show the disease in the same acute character as the European?

Sir RUBERT BOYCE replied that he had brought records of the cases of yellow fever occurring in the natives of West Africa this year. It would be found that the symptoms in these cases very closely resembled the bilious remittent fever symptoms to which he had drawn attention; that was to say, these symptoms were in every respect those of yellow fever, except that they stopped short of black vomit. The attacks were, therefore, not so severe as in the white; in fact they were distinctly less severe.

In Barbados the case was quite the contrary. The natives in Barbados suffered severely, and this was, he thought, as one would expect it to be, because the Barbadian native had been protected for many years. He had become non-immune, and therefore his attacks were very severe, like those of the Creole. Referring to the difference in the severity of the symptoms in case of natives of West Africa and of the West Indies respectively, Sir RUBERT BOYCE stated that the French and Brazilian investigators gave the following explanation:— One attack of yellow fever did confer a complete degree of immunity. Therefore, given a man who had had an attack of that disease in childhood (which would not have been diagnosed as such, because it was so mild); then again in youth and again in manhood, on each occasion he was becoming more immunised. When therefore he became infected with the virus side by side with the white man he had manifested all the symptoms of yellow fever, but in a much milder form than the white man by reason of his previous protection. Barbadian freedom from yellow fever in the past was quite a different thing to the immunity of the Sierra Leonian. In the former case it was due to protective measures, such as hygiene and controlled water supply. In the West African case it was due to the process of immunising. But when the Barbadian was exposed to imported infection as last year, 1909, he immediately got it.

He further stated that he believed that yellow fever could be absolutely stamped out by anti-stegomyia measures.

AUTOCTHONOUS ORIENTAL SORE IN PANAMA.

By Dr. S. T. DARLING,

Chief of Laboratory, Ancon Hospital, Canal Zone.

This account of an autocthonous case of Oriental sore in Panama is of interest, because it adds one other to the list of regions to be included in the geographical distribution of this disorder; and as the case is a solitary one, it raises again the question of the individuality or specificity of the different strains of *Leishmania-tropicum* causing Oriental sore. The lesion in this case apparently followed the bite of a tabanid.

Very few cases of Oriental sore have been reported from the New World. Wright's case, reported from Boston, Mass., U.S.A., from which the etiological factor was first described was an Armenian girl, and the lesion had appeared before she had left Armenia.

Juliano Moreira, in 1896, reported its occurrence in Bahia, Brazil.

Five cases were reported by Carini and Paranhos* among Syrian, Portuguese and Brazilian labourers at Baurú, St. Paul, Brazil.

A case has also been reported occurring in French Guiana.†

The case described here is the first that has been brought to the attention of the writer, who had searched among natives of this region and those of the West Indies for several years. The failure may be attributed partly to the fact that the hygienic surroundings of the Canal employees and of the inhabitants of the Canal Zone and of the cities of Colon and Panama have been vastly improved since the American occupation. Examples of Tropical ulceration, so common in 1904, and early in 1905, have almost entirely disappeared, except among those who are living out in the bush beyond the pale of sanitary measures.

The patient, J. A. C., negro, aged 44 years, was born in Demerara. He lived in Paramaribo three years and in Nickeri for 18 months, and subsequently in Trinidad for 24 years. In September, 1909, he came to the City of Panama, where he lived one month. Since October, 1909,

* Revista Medica de São Paulo, Brazil, March 31st, 1909.

† Nattan-Larrier, Tonin et Keckenroth : Bull. Soc. path. exot. p. 587, 1909.

he has been living on a cacao and rubber plantation, several miles from Empire, out in the bush. The residents at the plantation are native Colombians and Jamaican negroes. There are no natives of nor visitors from the Levant, India, or other regions of the Old World where Oriental sore is endemic. The patient states that he has never noticed anywhere a sore similar to his.

One day in May, of this year, while sitting under a wild cashew tree, the man was bitten on the forearm by a large fly, which, from his description, was a tabanid. The wound was scratched, and the resulting sore exuded a watery fluid. The patient, to relieve the itching, continually pinched and scratched the sore, thus preventing the formation of a crust. The sore remained in this condition for about a month, when it increased in size and a very hard thick crust formed on its surface. The itching now ceased, but the margins became quite tender. During the past three months the sore has been from 20 to 30 mm. in diameter.

The patient presented himself at the laboratory on August 17th, 1910. The sore was located on the right forearm near the wrist on the ulnar aspect. It was circular, and covered with a raised, flat, granular, greyish-yellow crust, which was very thick and hard. At this time, after four months' duration, the outside diameter, including the raised skin margin, was 22 mm.; the diameter of the crust was 17 mm. Upon lifting the crust a circular fossa was seen, separating the raised skin-margin from the granulating and healing surface of the ulcer. The appearance and location of the ulcer were so characteristic that the diagnosis of Oriental sore was at once suggested. Portions of the crust were removed and smears made from the granulating surface, which bled easily. A few micro-organisms, exactly resembling *L. tropicum*, were found. On August 26th the patient entered the hospital for treatment. The ulcer was excised well beyond the raised margins and below to normal subcutaneous tissue (Dr. HERRICK).

CHARACTERS OF THE MICRO-ORGANISMS.

Smears from its granulating surface contain free and intracellular micro-organisms, resembling *L. tropicum*. Most of them are oval, one end obtuse, the other more acute. All contain a tropho-nucleus and a kineto-nucleus; a few present small achromatic spaces in the pale blue

cytoplasm. A refractile capsule is not detected; some are in intracellular groups of two, four, five, and eleven, or more.

DIMENSIONS OF THE MICRO-ORGANISMS.

Length.	Breadth.	
6 mm.	3·5 mm.	
5 mm.	3·5 mm.	Length of kineto-nucleus 1·5 mm.
6 mm.	3·0 mm.	Diameter of tropho-nucleus 2·75 mm.
9 mm.	4·25 mm.	

The micro-organism, while slightly larger than some of those described elsewhere, presents the same morphology noted in cases from the Old World, and it closely resembles the gregarine phase of *Crithidia* found in representatives of *Tabanus* and other invertebrates. It is not impossible that the case described here, following as it does the history of a bite by a tabanid, is the result of an inoculation with an invertebrate gut flagellate *(Crithidia ?)*, which was able to take on a parasitic existence in the tissues of man.

HISTOLOGICAL EXAMINATION OF THE TISSUE FROM THE ULCER.

The corium and papillæ are richly infiltrated by newly-formed cells of the lymphoid, endothelial, plasma, epithelioid, and giant cell types, the latter having a mural arrangement of the nuclei. There is no polymorphonuclear leucocytic infiltration and no necrosis. The surface of the ulcer is nearly covered with squamous epithelium which shows considerable metaplasia. Numerous interpapillary down growths of the rete are seen, sometimes enclosing a cell-nest and separating the newly-formed cells in the corium into parallel, oblong, and circular chambers. The squamous epithelium is surmounted by a mass of desquamated epithelium (crust), staining intensely with eosin. The sweat glands, lymphatic and blood vessels in the deeper layers of the skin, are almost always surrounded by newly-formed collections of small, round cells of the lymphoid type. A few intracellular micro-organisms, resembling *L. tropicum*, are seen singly or in small groups alongside the nucleus of an endothelial epithelioid or other cell in the corium. One micro-organism was seen close beside the margin of a giant cell.

Smears from the ulcer were stained for the presence of acid fast bacilli, but none were found.

This brief description of the histopathology of the ulcer, while differing from that of WRIGHT, is not unlike, in certain particulars, those of THOMPSON* and BALFOUR and FIRTH.†

This is probably due to the fact that descriptions in the literature have been made at different stages in the development of the lesion—*i.e.*, the predominance of parasitised endothelial cells and atrophy of the epithelium being seen earlier, while the predominance of lymphoid and plasma cells, the appearance of the epithelioid and giant cells with metaplasia of epithelium, and relatively fewer micro-organisms, being noted in the older lesions; or the differing histological pictures may be the result of infections by different varieties of flagellates having different degrees of virulence, and eliciting different histological changes.

The case is very clearly one of Oriental sore—a term, however, which should, no doubt, be replaced by one more definitely related to the pathogenic agent and the location of the lesion, such as Dermal Leishmaniosis or Ulcerating Leishmaniosis.

The lesion occurred in a West India negro, native of Demerara, who had been living on the Isthmus of Panama eight months. It followed the bite of a fly, which was evidently a tabanid.

The pathogenic agent, *L. tropicum*, was found in small numbers, free and intracellular, in smears and sections of tissue. It was somewhat larger than those described from the Old World, and possibly represents a distinct species.

The symptomatology, location of lesion, and histopathology correspond with those of Oriental sore.

* Transactions of the Society of Tropical Medicine and Hygiene. London, Vol. 3, No. 3, pp. 107-128.

† Allbutt's System of Medicine.

ELECTION OF MEMBERS.

The following were duly elected as Fellows of the Society :—

David Alexander, M.B., Northern Nigeria.

Johan Cammermeyer, M.D., Chief of the Congo Medical Service.

Andrew Graham, L.R.C.P., Ichang.

J. Hormusji, L.R.C.P., Rangoon.

Mahomed Kamel, Cairo, Egypt.

J. MacFarlane Pollard, M.B., West African Medical Staff.

J. C. PHELP AND SON, PRINTERS, WALTHAMSTOW.